GUIDELINE for Bordetella bronchiseptica infection in cats

Published: 01/01/2009
Last updated: 08/06/2022
Last reviewed: 09/02/2024

These Guidelines were first published by Herman Egberink et al. in Journal of Feline Medicine and Surgery 11 (7), 2009, 610-614. This update has been compiled by Herman Egberink.

Key points

- *Bordetella bronchiseptica* (*Bb*) is a primary pathogen of cats.
- The bacterium is shed in oral and nasal secretions of infected cats.
- The bacterium is susceptible to common disinfectants.
- Cats can catch *Bb* infection from dogs with kennel cough.
- *Bb* colonises the ciliated epithelium of the respiratory tract of the host.
- A wide range of respiratory signs has been associated with *Bb* infection, from mild upper respiratory signs to those of severe pneumonia with dyspnoea, cyanosis and potentially death, especially in young kittens.
- When sensitivity data are unavailable, tetracycline antibiotics are recommended, since most feline *Bb* isolates are susceptible.
- In some European countries an intranasal modified live vaccine is available.
- Zoonotic infections have been incidentally recorded in the literature.

Agent properties

Bordetella bronchiseptica (*Bb*) is a primary respiratory pathogen of cats, particularly in high population density conditions such as rescue shelters and multicat households.

Bordetella (*B.*) *pertussis*, *B. parapertussis* and *Bb* are closely-related Gram-negative coccobacilli that colonise the respiratory tracts of mammals. *B. pertussis* is a strictly human pathogen and the primary aetiologic agent of whooping cough, which can also be caused by a human lineage of *B. parapertussis*. Being the least host-restricted member of this taxonomic cluster, *Bb* can cause chronic respiratory infections in cats, dogs, rabbits, pigs and rarely humans. Sequence analysis has shown that *B. parapertussis* and *B. pertussis* are independent derivatives of *Bb*-like ancestors. During their evolution, there was large-scale gene loss and inactivation; host adaptation seems to be a consequence of loss, not gain, of function, and differences in virulence could be related to loss of regulatory or control functions (Parkhill et al., 2003).

Epidemiology

Prevalence

Infection with *Bb* is widespread in the cat population as evidenced by the high level of seroprevalence and isolation rates from cats with and without respiratory disease (Gaskell et al., 2011). The larger the group of cats is, the higher is the prevalence of *Bb* infections.

In a large survey of pathogens associated with respiratory disease in multicat (≥5) households in nine European countries, *Bb* was

detected by PCR in 5 % of cats from households with signs of respiratory disease and in 1.3 % of households in which no cat showed respiratory disease (Helps et al., 2005). The larger the group, the more likely a cat was to be found infected. PCR will have underestimated the true prevalence since in this study it was found to be less sensitive than bacterial culture. In a cross-sectional survey of a convenience sample (740 cats), *Bb* was isolated in 19 % of cats in rescue catteries, in 13.5 % in research colonies and not at all in household pets (Binns et al., 1999). In a study of clinical field cases of cats with upper respiratory tract disease (URTD) *Bb* was isolated from only 0.4% of 460 samples (Adler et al., 2007). In another survey, only 3/52 cats with acute respiratory signs living in a shelter were shown to be *Bb*-culture positive (Veir et al., 2008). A similar low prevalence was determined in a more recent study in a shelter in which 4/82 cats with upper respiratory tract clinical signs were shown to be *Bb* culture positive (Walter et al., 2020).

Antibody prevalence was 61 % and 41 % in household with *versus* without respiratory signs, respectively. Poor hygiene in rescue shelters was associated with higher antibody prevalence (Helps et al., 2005). In another antibody prevalence study in multicat households, a significant difference was found in antibody prevalence between cats with and without clinical signs of URTD. Also, especially in animal shelters, the risk of being seropositive was shown to be high (Gaston et al., 2005).

Transmission

The bacterium is shed in oral and nasal secretions of infected cats (Speakman et al., 1999). After experimental infection, the organism was isolated in some cases for at least 19 weeks (Coutts et al., 1996).

Direct and indirect contacts with such secretions are probably mainly responsible for *Bb* transmission, although this has not been experimentally confirmed. As with other respiratory pathogens, such as feline calicivirus (FCV) and feline herpesvirus (FHV), overcrowding and poor management predispose to infection and disease.

Dogs with respiratory disease are a risk factor for cats; dog-to-cat transmission of *Bb* has been confirmed by molecular data (Dawson et al., 2000).

Bb was also cultured from post-parturient queens that had been culture-negative previously. Under these conditions, the kittens remained *Bb* antibody-negative (Coutts et al, 1996).

The physicochemical stability of *Bb* is unknown. The mean environmental persistence of *B. pertussis* is longer than 10 days, and *Bb* probably is equally hardy, so indirect transmission must be assumed (Walther and Ewald, 2004). The bacterium is susceptible to common disinfectants.

Pathogenesis

Bb is a primary pathogen for cats. Respiratory disease has been reproduced in specific pathogen-free cats after aerosol and nasal challenge (Willoughby et al., 1991; Jacobs et al., 1993; Coutts et al., 1996; Welsh, 1996). Infections occur frequently in the field, but several factors are involved in disease development, including environmental conditions leading to stress (e.g. overcrowding) and/ or pre-existing viral infections.

Little information exists about the true pathogenicity of *Bb* as a single infection in the cat, and much has to be inferred from infections in other species. Features responsible for *Bb* acting as a primary pathogen in the feline respiratory tract are its motility (propulsion by flagella), the presence of adhesins and toxin production (Gaskell et al., 2011).

This microorganism colonises the ciliated epithelium of the respiratory tract of the host establishing chronic infections. *Bordetellae* have evolved mechanisms, some of them shared, that allow them to colonise this site, a surface designed to eliminate foreign particles (Mattoo et al., 2001). These include adhesins, such as filamentous hemagglutinin, fimbriae and periactin. Fimbriae are required for efficient and persistent colonisation of the trachea. They also play an important role in the development of humoral immunity to *Bb* infection (Mattoo et al., 2000). Once attached, toxins and *Bb*-specific secreted proteins result in ciliostasis and destruction of the cilia.

Immunity

Antibodies play an important role in the immune response to *Bb* and bacterial clearance. Cats vaccinated with modified live intranasal FHV and FCV vaccines showed reduced clinical signs during the first ten days after *Bb* challenge infection. One study showed that a specific innate immunity could also be beneficial for short term protection (Bradley et al., 2012).

Passive immunity

Little information is available on the transmission of maternally derived antibodies (MDA) to kittens. In one study of kittens born to *Bb* antibody-positive queens, MDA remained low and were only detectable for 2 weeks (Coutts et al., 1996). In another study, low levels of MDA remained detectable for 8 weeks but were not assayed for longer (Jacobs et al., 1993).

Active immune response

After *Bb* infection, serum antibodies rise rapidly, but it is unknown for how long they persist (Coutts et al., 1996). Immunoglobulin A (IgA) is the main class in mucosal secretions. Individuals deficient in IgA are more susceptible to certain infections of the paranasal sinuses and airways of the lung (sinopulmonary infections) (Renegar et al., 2004). In mice it was shown that IgA is also essential for controlling *Bb* in the upper respiratory tract. Transfer of IgA-containing convalescent serum effectively reduced *Bb* numbers in the trachea; they cleared only *Bb*, but not human *Bordetella* pathogens (Wolfe et al., 2007).

Clinical signs

Experimental infection of specific pathogen-free cats induced mild clinical signs consisting of fever, coughing, sneezing, ocular discharge and lymphadenopathy, which resolved after about 10 days (Jacobs et al., 1993; Coutts et al., 1996).

In the field, a wide range of respiratory signs has been associated with *Bb* infection, from mild clinical signs, such as coughing, sneezing, ocular discharge, fever, and lymphadenopathy to severe pneumonia with dyspnoea, cyanosis and potentially death (Willoughby et al., 1991; Welsh, 1996; Speakman et al., 1999). Pneumonia is usually seen in kittens of less than 12 weeks, but older cats can be affected as well (Figs. 1, 2 and 3). *Bb* infection should be considered as a differential in cats with acute and chronic cough.

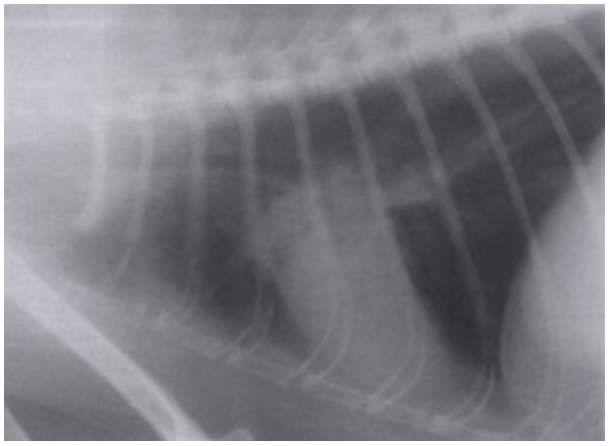

Fig. 1. Pneumonia caused by Bordetella bronchiseptica infection. Courtesy of Andy Sparkes, UK

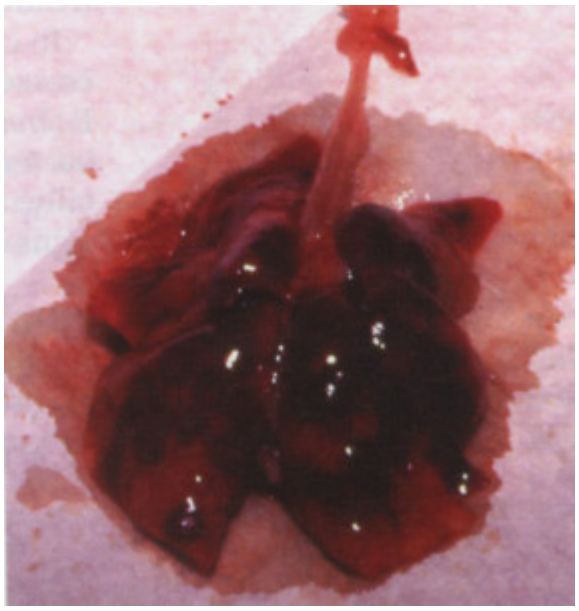

Fig. 2. Lungs of a kitten that had died of pneumonia. Bordetella bronchiseptica was isolated from the organ. Courtesy of Maria Grazia Pennisi, University of Messina, Italy

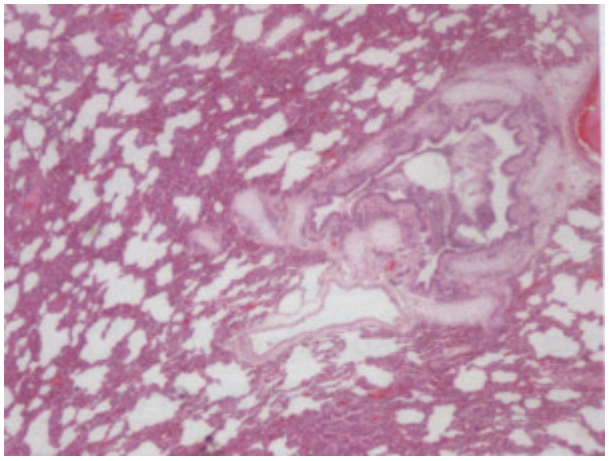

Fig. 3. Lungs section from a kitten that had died of pneumonia. Bordetella bronchiseptica was isolated from the organ. Courtesy of The Feline Centre, University of Bristol, UK

Diagnosis

In cats infected with *Bb*, cytological analysis of tracheal washes can demonstrate polymorphonuclear leucocytes, macrophages and bacteria (Welsh, 1996).

Direct detection of the infectious agent

For direct detection of the bacterium or bacterial DNA, both bacterial culture (isolation) and PCR are available. Samples for culture can be obtained from oropharyngeal swabs, nasal cavity swabs or broncheo-alveolar lavage samples. Transtracheal washes as a source for culture are also described in one study (Welsh, 1996). In one study, nasal swabs were shown to be more often culture-positive than oropharyngeal swabs, suggesting that for *Bb* isolation nasal swab is preferred over an oropharyngeal swab (Veir et al. 2008). However, the significance of *Bb* in nasal, oropharyngeal, or nasopharyngeal swabs is questionable as it may not represent a primary pathogen but rather be opportunistic here instead.

Bacterial culture

For bacterial culture (isolation), swabs should be placed into charcoal (or regular) Amies transport medium. *Bb* should be cultured on a selective medium, such as charcoal/cephalexin agar, which reduces overgrowth by other respiratory bacteria. If *Bb* is identified from bronchoalveolar lavage samples of cats with lower respiratory signs an etiological role is assumed. The significance of Bordetella in oropharyngeal swabs from cats with predominantly upper respiratory clinical signs is less clear, as outlined above; it may be that antibiotic treatment is indicated if no other underlying causes are found.

In cats from multicat households and other crowded environments, the prevalence of *Bb* infection is higher than in other conditions and the bacterium is often present co-incidentally in the upper respiratory tract (Gaskell et al., 2011); other causes for the presenting clinical signs must then be considered (Gaston et al., 2005).

PCR

Sensitive real-time PCR methods are capable of discriminating between different *Bordetellae* and can detect less than 10 genome

copies of *Bb*/µl (Koidl et al., 2007). Some laboratories have developed multiplex assays that allow the simultaneous detection of several common feline respiratory pathogens. In one study, *Bb* (and FHV and *Mycoplasma felis*) was found more frequently using real-time PCR than bacterial culture (Litster et al., 2015), although differences were not significant due to the low number of samples included.

Indirect detection of the infectious agent

Antibody detection is of limited diagnostic use due to the high antibody prevalence in the general cat population (Gaskell et al., 2011).

Treatment

Antimicrobial treatment

Antibacterial therapy is indicated if *Bb* infection is considered the cause of clinical signs, even when the signs are mild, because *Bb* might progress to colonise the lower respiratory tract. Therapy should be based on the results of antibiotic susceptibility testing. In a study with 42 canine or feline *Bb* strains, a considerable number of strains were shown to be resistant or exhibited high minimum inhibitory concentration (MIC) values against a number of antibacterial agents (Schwarz et al., 2007). Where sensitivity data are unavailable, doxycycline is recommended as the antimicrobial of choice, since most feline *Bb* isolates are susceptible. Oral doxycycline (5 mg/kg q12h or 10 mg/kg q 24h) for 7-10 days is recommended as a first line treatment in the ISCAID guidelines (Lappin et al., 2017). Different formulations of doxycycline exist but it should be noted that doxycycline hydrate tablets can cause oesophagitis if incompletely swallowed, so dosing of this formulation, if used, should be followed by food and/or water to encourage complete swallowing and passage of the tablet into the stomach.

Feline *Bb* isolates have low susceptibility to clavulanate-potentiated amoxycillin, and resistance has often been detected to ampicillin and trimethoprim (Speakman et al., 1997). Whilst antimicrobial therapy should help to alleviate clinical signs, antibiotic treatment in recovered carrier cats has little effect on shedding (Coutts et al., 1996).

Symptomatic treatment

Cats severely affected by *Bb* infection require additional supportive therapy and intensive nursing care. The resolution of dehydration and restoration of electrolyte and acid-base disturbances preferably by intravenous fluid administration can be needed for cats with severe clinical signs. With severe lower respiratory disease and dyspnoea, oxygen therapy should be considered.

If the cats do not eat, appetite stimulants (e.g. mirtazapine; 1.88-2 mg/cat PO, q24h if renal and hepatic function is not compromised) can be indicated. Alternatively, commercial fluid high-energy diets can be used for hand feeding. If the cat is not eating for more than three days, placement of a feeding tube and enteral nutrition is indicated.

In cats with pneumonia, nebulization can help to rehydrate the upper respiratory tract, loosen secretions, reduce congestion and increase comfort. Cheap small nebulisers can be purchased for use in the veterinary clinic (or loaned to owners to use at home) for regular nebulization therapy with saline (q 4-6 hours if possible for 15 mins at a time). In the hospital setting this can be applied close to the cat's face by placing the cat in an igloo and covering the exit and nebulizing inside the igloo. If a nebuliser is not available, an alternative is to provide steam therapy. Owners can do this at home by sitting with their cat with care in a bathroom with hot running water/shower. Drugs with mucolytic effects (e.g. bromhexine) may be helpful.

Vaccination

General recommendations on vaccination

In some European countries an intranasal modified live vaccine is available. The ABCD does not recommend routine vaccination against *Bb* (non-core), since the infection generally causes only mild disease. Vaccination should be limited to cats living in or moving into high-density populations with a history of *Bb* infections and should be performed according to the manufacturer's recommendations.

Bb vaccines containing viable bacteria should never be administered to kittens less than 4 weeks of age as recommended by the manufacturer. In addition, they are ineffective in cats on, or due to receive, antibiotics. Cats receiving live vaccines will shed bacteria, and these vaccines must be avoided where an owner is known to be immunocompromised (Moore et al., 2021). These vaccines can occasionally induce mild clinical signs.

Since the *Bb* vaccine contains live bacteria there is a potential risk of infection during administration of the vaccine or indirectly through contact with a vaccinated cat during the post vaccination shedding period (Moore et al., 2021). As stated in the SPC, the shedding period can last up to one year. Therefore, the SPC includes the following precaution: "although the risk of immunocompromised humans becoming infected with *Bb* is extremely low, it is advised that cats, which are in close contact with immunocompromised humans are not vaccinated with this vaccine."

Primary vaccination course

The modified live vaccine is licensed for use as a single vaccination with annual boosters.

Booster vaccinations

Annual boosters are recommended if protection should be maintained. Duration of immunity of at least a year has been demonstrated (Williams et al., 2002). Boosters are preferably given prior to going into a boarding or rescue cattery which has a history of *Bb* infections or before going to a cat show. Boosters should be continued as long as the cat remains in the high-risk situation.

Disease control in specific situations

Control of *Bb* in cat populations is aimed at minimising the exposure of naïve cats. Stocking densities needs to be reduced and the environment cleaned and disinfected to minimize the risk of transmission. Otherwise, the measures advocated for the control of other common respiratory pathogens, such as FCV and FHV, in groups of cats will also help control *Bb* infection and disease.

Shelters

Random source populations with largely unknown vaccination histories, continuous resident turnover, and high risk for infectious disease characterize most shelters. In these, *Bb* vaccination is encouraged, particularly if there is a history of confirmed *Bb*-associated disease.

Breeding catteries

The vaccination schedules used for privately owned cats are appropriate for most breeding catteries. *Bb* vaccination is rarely indicated in breeding catteries.

See also the ABCD Tool "Vaccine recommendations for cats according to their lifestyle".

Vaccination of immunocompromised cats

Vaccination of immunocompromised cats is not recommended. See also the ABCD guidelines on vaccination of immunocompromised cats.

Zoonotic risk

Because of its role as a potential human pathogen, *Bb* is important (Woolfrey and Moody, 1991; Bauwens et al., 1992) although most cases in humans occur in immunocompromised patients, without clear evidence for exposure to animals. Zoonotic infections have been incidentally recorded in the literature: a possible human infection from a rabbit, infections in paediatric lung transplant recipients, where dogs had been suspected as the origin of infection, and cases in immune compromised humans where cats were suspected as source of the infection (Gueirard et al., 1995; Ner et al., 2003; Redelman-Sidi et al., 2011; Wernli et al. 2011). It is sensible therefore to consider *Bb* as a rare potential cause of zoonotic infections.

Acknowledgement

ABCD Europe gratefully acknowledges the support of Boehringer Ingelheim (the founding sponsor of the ABCD), Virbac, IDEXX GmbH and MSD Animal Health.

References

Adler K, Radeloff I, Stephan B, Greife H, Helelmann K (2007): Bacteriological and virological status in upper respiratory tract infections of cats. Berl.Munch.Tierarztl.Wochenschr. 120 (3-4), 120-125.

Bradley A, et al (2012): Efficacy of Intranasal Administration of a Modified Live Feline Herpesvirus 1 and Feline Calicivirus Vaccine against Disease Caused by Bordetella bronchiseptica after Experimental Challenge. J Vet Intern Med 26(5), 1121-1125.

Bauwens JE, Spach DH, Schacker TW, Mustafa MM, Bowden RA (1992): Bordetella bronchiseptica pneumonia and bacteremia following bone marrow transplantation. J Clin Microbiol 30, 2474-2475.

Binns SH, Dawson S, Speakman AJ, et al (1999): Prevalence and risk factors or feline Bordetella bronchiseptica infection. Vet Rec 144, 575-580.

Coutts AJ, Dawson S, Binns S, Hart CA, Gaskell CJ, Gaskell RM (1996): Studies on natural transmission of Bordetella bronchiseptica in cats. Vet Microbiol 48, 19-27.

Dawson S, Gaskell CJ, McCracken CM, Gaskell RM, Hart CA, Jones D (2000): Bordetella bronchiseptica infection in cats following contact with infected dogs. Vet Rec 146, 46-48.

Gaskell RM, Dawson S, Radford A (2011): Feline respiratory disease. In: Green CE, ed. Infectious diseases of the dog and cat. St Louis: Saunders Elsevier, 151–161.

Gastón JZ, et al (2005): Prevalence of antibodies against Bordetella bronchiseptica in multi-cat households. Tierarztliche Praxis Ausgabe K: Kleintiere – Heimtiere 33(3), 197-201.

Gueirard P, Weber C, Le Coustumier A, Guiso N (1995): Human Bordetella bronchiseptica infection related to contact with infected animals: persistence of bacteria in host. J Clin Microbiol 33, 2002-2006.

Helps CR, Lait P, Damhuis A, Björnehammar U, et al (2005): Factors associated with upper respiratory tract disease caused by feline herpesvirus, feline calicivirus, *Chlamydophila felis* and *Bordetella bronchiseptica* in cats: experience from 218 European catteries. Vet Rec 156, 669-673.

Jacobs AA, Chalmers WS, Pasman J, van Vugt F, Cuenen LH (1993): Feline bordetellosis: challenge and vaccine studies. Vet Rec 133, 260-263.

Koidl C, Bozic M, Burmeister A, Hess M, Marth E, Kessler HH (2007): Detection and differentiation of Bordetella spp. by real-time PCR. J Clin Microbiol 45, 347-350.

Lappin MR, Blondeau J, Boothe D, Breitschwerdt EB, Guardabassi L, Lloyd DH, Papich MG, Rankin SC, Sykes JE, Turnidge J, Weese JS (2017): Antimicrobial use Guidelines for Treatment of Respiratory Tract Disease in Dogs and Cats: Antimicrobial Guidelines Working Group of the International Society for Companion Animal Infectious Diseases. J Vet Intern Med31(2), 279-294. doi: 10.1111/jvim.14627

Litster A, Wu CC, Leutenegger CM (2015): Detection of feline upper respiratory tract disease pathogens using a commercially available real-time PCR test. Vet J 206(2), 149-153.

Mattoo S, Foreman-Wykert AK, Cotter PA, Miller JF (2001): Mechanisms of Bordetella pathogenesis. Front Biosci 6, 168-186.

Mattoo S, Miller JF, Cotter PA (2000): Role of *Bordetella bronchiseptica* fimbriae in tracheal colonization and development of a humoral immune response. Infect Immun 68, 2024–2033.

Moore JE, Rendall JC, Millar BC (2021): A doggy tale: Risk of zoonotic infection with *Bordetella bronchiseptica* for cystic fibrosis (CF) patients from live licenced bacterial veterinary vaccines for cats and dogs. J Clin Pharm Ther 2021;00:1–7. Doi. 10.1111/jcpt.13492

Ner Z, Ross LA, Keens TG, MacLaughlin EF, Starnes VA, Woo MS (2003): *Bordetella bronchiseptica* infection in pediatric lung transplant recipients. Pediatr Transplant 7, 413-417.

Parkhill J, Sebaihia M, Preston A, et al (2003): Comparative analysis of the genome sequences of *Bordetella pertussis, Bordetella parapertussis* and *Bordetella bronchisepti*ca. Nat Genet 35, 32-40.

Redelman-Sidi G, Grommes Ch, Papanicolaou G (2011): Kitten transmitted *Bordetella bronchiseptica* infection in a patient receiving temozolomide for glioblastoma. J Neurooncol 102, 335-339.

Renegar KB, Small PA Jr., Boykins LG, Wright PF (2004): Role of IgA versus IgG in the control of influenza viral infections in the murine respiratory tract. J Immunol 173, 1978-1986.

Schwarz S, Alesik E, Grobbel M, Lübke-Becker A, Werckenthin C, Wieler LH, et al (2007): Antimicrobial susceptibility of Pasteurella multocida and Bordetella bronchiseptica from dogs and cats as determined in the BfT-GermVet monitoring program 2004-2006. Berl MunchTierarztlWochenschr 120(9-10), 423-430.

Speakman AJ, Binns SH, Dawson S, Hart CA, Gaskell RM (1997): Antimicrobial susceptibility of *Bordetella bronchiseptica* isolates from cats and a comparison of the agar dilution and E-test methods. Vet Microbiol 54, 63-72.

Speakman AJ, Dawson S, Binns SH, Gaskell CJ, Hart CA, Gaskell RM (1999): *Bordetella bronchiseptica* infection in the cat. J Small Anim Pract 40, 252-256.

Veir JK, Ruch-Gallie R, Spindel ME, Lappin MR (2008): Prevalence of selected infectious organisms and comparisons of two anatomic sampling sites in shelter cats with upper respiratory tract diseases. J Feline Med Surg 10(6), 551-557.

Walter J, Foley P, Yason C, Vanderstichel R, Muckle A (2020): Prevalence of feline herpesvirus-1, feline calicivirus, Chlamydia felis, and Bordetella bronchiseptica in a population of shelter cats on Prince Edward Island. Canadian Journal of Veterinary Research-Revue Canadienne De Recherche Veterinaire 84(3), 181-188.

Walther BA, Ewald PW (2004): Pathogen survival in the external environment and the evolution of virulence. Biol Rev Camb Philos Soc 79, 849-869.

Welsh RD (1996): *Bordetella bronchiseptica* infections in cats. J Am Anim Hosp Assoc 32, 153-158.

Wernli D, Emonet S, Schrenzel J, Harbarth S (2011): Evaluation of eight cases of confirmed *Bordetella bronchiseptica* infection and colonization over a 15-year period. Clin Microbiol Infect 17(2), 201-203.

Williams J, Laris R, Gray AW, Jacobs AA (2002): Studies of the efficacy of a novel intranasal vaccine against feline bordetellosis. Vet Rec 150: 439-442.

Willoughby K, Dawson S, Jones RC, et al (1991): Isolation of B. bronchiseptica from kittens with pneumonia in a breeding cattery. Vet Rec 129, 407-408.

Wolfe DN, Kirimanjeswara GS, Goebel EM, Harvill ET (2007): Comparative role of immunoglobulin A in protective immunity against the Bordetellae. Infect Immun 75, 4416-4422.

Woolfrey BF, Moody JA (1991): Human infections associated with *Bordetella bronchiseptica*. Clin Microbiol Rev 4, 243-255.